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Office of Administrative Law Judges
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Issue Date: 14 September 2007

Case No.: **2004-BLA-00153**

In the Matter of:

L. W. (widow of J. W.),
Claimant,

v.

CANYON FUEL COAL LLC,
(SOLDIER CREEK COAL COMPANY)
c/o ARCH COAL COMPANY,
Employer,

COASTAL STATES ENERGY COMPANY,
Carrier,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-In-Interest.

DECISION AND ORDER ON REMAND

This case arose from a claim for federal benefits under the "Black Lung Benefits Act," Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 et seq. ("ACT"), and applicable regulations, mainly 20 C.F.R. Parts 410, 718 and 727 ("Regulations").

The Act and Regulations provide compensation and other benefits to: (1) living coal miners who are totally disabled due to pneumoconiosis and their dependents; (2) surviving dependents of coal miners whose death was due to pneumoconiosis; and (3) surviving dependents of coal miners who were totally disabled due to pneumoconiosis at the time of their death (for claims filed prior to January 1, 1982). See also Sections 718.306 and 727.204 for entitlement presumptions in certain claims filed before April 30, 1982 where the miner was partially disabled at death. Other benefits include necessary medical and hospitalization costs required for the treatment of pneumoconiosis. The Act and Regulations define pneumoconiosis ("black lung disease" or "CWP") as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. See 20 C.F.R. § 718.201 (2003).

Procedural History

On July 29, 1992, the Miner filed a claim for benefits. (DX 28) [1] The District Director denied this application on December 17, 1992. (DX 28) On March 1, 1999, the Miner filed a second application for benefits. (DX 1) Before a Decision and Order could be issued in that claim, the Miner died. (DX 4) His death occurred on June 4, 1999. (DX 4) On August 31, 1999, the Miner's widow filed a claim on her own behalf. (DX 29) A Decision and Order consolidating those claims and awarding benefits in both claims was issued on November 6, 2001 by Administrative Law Judge Paul H. Teitler. (DX 84) The Employer timely appealed the administrative law judge's decision to the Benefits Review Board (the Board). The Board affirmed the Decision and Order awarding benefits in both claims on October 24, 2002. (DX 100) The Board denied the Employer's Motion for Reconsideration. (DX 106)

The Employer then filed a timely request for modification on May 9, 2003. (DX 110) The District Director denied the request for modification. (DX 119, DX 121) The Employer appealed this decision and the case was assigned to the undersigned on January 5, 2005. (DX 122) The Claimant then filed a Motion to Dismiss the Modification Petition, arguing that the Employer was not a party and that the Modification Petition was not timely as to the living miner's claim. The undersigned issued a Decision and Order in this case on November 8, 2005. In this order, the undersigned denied the Claimant's Motion to Dismiss and denied the Employer's petition for modification. The Claimant's benefits would therefore continue as previously awarded.

Both parties appealed this decision. The Claimant argued on appeal that the administrative law judge erred in considering the Employer's petition for modification without compelling the Employer to allege specific factual errors in the award of benefits. The Claimant also argued that the administrative law judge neglected to determine whether altering the award of benefits in either claim would serve the principles of equity and fairness. Finally, the Claimant alleged that the burden of proof was erroneously placed upon her to establish entitlement when the burden of proof should have been placed on the Employer, since the Employer was the proponent of the petition for modification. The Employer argued on appeal that the administrative law judge did not provide a valid rationale for discrediting the opinions of three doctors, Dr. Farney, Dr. Tuteur and Dr. Tomashefski, who stated that pneumoconiosis was not a contributing cause of the Miner's pulmonary impairment and that pneumoconiosis did not hasten the Miner's death.

In its Decision dated September 28, 2006, the Board stated that "the administrative law judge did not err in declining to require employer to allege specific errors of fact in the prior

[1] The following abbreviations will be used as citations to the record:
ALJX – Administrative Law Judge's Exhibits;
EX – Employer's Exhibits;
DX – Director's Exhibits;
CX – Claimant's Exhibits; and
TR – Transcript of the Hearing.

denial. As claimant herself has noted, the Board has held that the contents of a petition for modification need not comply with any formal requirements…."

The Board further stated that

> Claimant has indicated correctly, however, that the administrative law judge's analysis of employer's petition for modification was not complete. Claimant asserts that the administrative law judge neglected to determine whether altering the award of benefits in either claim would serve the principles of equity and fairness. Although not stated in precise terms, claimant's argument has merit, inasmuch as the administrative law judge did not include a consideration of whether reopening the award of benefits would render justice under the Act.

The Board also noted that the administrative law judge "must place the burden of proof on employer when assessing whether the prior decision awarding benefits contains a mistake in determination of fact pursuant to Section 725.310 (2000)."

Finally, the Board also determined that, while the administrative law judge acted within his discretion when according little weight to Dr. Farney's opinion, since Dr. Farney failed to acknowledge that the Miner suffered from hypoxemia, the administrative law judge did not provide adequate reasons for discrediting the reports of Dr. Tuteur and Dr. Tomashefski. The Board stated that

> [c]ontrary to the administrative law judge's findings … both Drs. Tomashefski and Tuteur discussed the results of the miner's 1999 post-exercise BGS and indicated that pneumoconiosis was not the source of the oxygen desaturation demonstrated by the study. Employer is also correct in asserting that the administrative law judge did not adequately explain how the fact that Drs. Tuteur and Tomashefski did not specifically address the abnormalities seen on the x-rays read as negative for pneumoconiosis detracted from the credibility of their opinions regarding the cause of the miner's total disability and death. Both physicians diagnosed simple pneumoconiosis based upon the miner's autopsy results and indicated that the negative x-ray readings were consistent with their determination that the miner's pneumoconiosis was mild.

On September 28, 2006, the Board remanded the case to the undersigned. The Board stated that on remand, the administrative law judge "must determine whether reopening the claims would render justice under the Act." Moreover,

> the administrative law judge must perform an independent assessment of the newly submitted medical opinion evidence, considered in conjunction with the previously submitted evidence, to determine if the weight of the new evidence is sufficient to establish that an error was made in the determination that the miner was totally disabled due to pneumoconiosis at the time of his death pursuant to Section 718.204(c) and that his death was due to pneumoconiosis pursuant to Section 718.205(c).

The burden of proof must be placed on the Employer when rendering this decision.

Evidence

Section 22 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 922, as incorporated into the Black Lung Benefits Act by 30 U.S.C. § 932(a), and as implemented at 20 C.F.R. § 725.310 (2000), provides that within one year of the last payment of compensation or rejection of a claim, a party-in-interest may request modification of a compensation award due to a mistake of fact or a change in condition. Prior case law has established that the finder of fact has "broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." O'Keeffe v. Aerojet-General Shipyards, 404 U.S. 254 (1971). Therefore, the undersigned will now perform an independent assessment of the newly submitted medical opinion evidence, considered in conjunction with the previously submitted evidence, to determine if the weight of the new evidence is sufficient to establish that an error was made in the determination that the miner was totally disabled due to pneumoconiosis and that his death was hastened by pneumoconiosis.[2]

New Evidence

Dr. Peter Tuteur

Dr. Peter Tuteur prepared a medical report dated May 5, 2003. (DX 113) Dr. Tuteur relied on medical reports from other physicians, an autopsy report, chest x-rays, arterial blood gas studies, pulmonary function tests, the Miner's death certificate, pulmonary pathology consultation reports, and a forensic pathologist's report. (DX 113)

Dr. Tuteur noted that the Miner worked in the underground mining industry for thirty-nine years, prior to his retirement in 1992 due to back and knee pain. (DX 113) Fourteen of those thirty-nine years were spent working "in a coal mine drilling and blasting rock." (DX 113) While working, the Miner occasionally experienced wheezing and a cough, which included yellow sputum and streaks of blood. (DX 113)

At the time of his retirement, "he was able to walk on flat ground but not as well as he had earlier in his life." (DX 113) After his retirement, the wheezing decreased and the cough

[2] When the Employer requested a modification, it claimed that a mistake of fact had been made. The Employer submitted new medical reports, by Dr. Farney, Dr. Tuteur and Dr. Tomashefski, and argued that the cumulative evidence proved that the Miner was not totally disabled due to pneumoconiosis at the time of his death and that the Miner's death was not hastened by his pneumoconiosis. The Employer never disputed that the Miner suffered from pneumoconiosis. Nor did the Employer claim that the Miner's pneumoconiosis could be attributed to a cause other than coal mine employment. Thus, since pneumoconiosis is not at issue at this time, the evidence used in prior cases to establish that the Miner had pneumoconiosis and that his pneumoconiosis arose out of coal mine employment will not be outlined in the present Decision and Order. Moreover, the parties seem to agree that the Miner was totally disabled prior to his death. As is noted above, at the present time, the only issues concern whether the Miner's total disability was due to pneumoconiosis and whether the Miner's death was due to pneumoconiosis. Thus, only evidence relating to those issues will be discussed in detail.

stopped. (DX 113) However, "breathlessness became much more prominent such that minimal exertion was not tolerated." (DX 113) Medical records suggested that the Miner's breathlessness during exertion was due, at least in part, to impairment in gas exchange. (DX 113) Although Dr. Tuteur stated that "the data set does not allow for a robust identification of the cause of the physiologic impairment of gas exchange during exercise," Dr. Tuteur eliminated coal workers' pneumoconiosis as a possible cause of this impairment. (DX 113)

The Miner died on June 4, 1999. (DX 113) Dr. Tuteur noted that an autopsy showed advanced coronary artery disease with 80% occlusion of two major branches and a complete 100% acute occlusion of the circumflex coronary artery. (DX 113) This caused an acute myocardial infarction, which caused the Miner's death. (DX 113) The autopsy also showed that the Miner had simple coal workers' pneumoconiosis and mild centrilobular emphysema. (DX 113)

Dr. Tuteur did believe that the Miner was totally disabled prior to his death. (DX 113) However, after examining the arterial blood gas tests, pulmonary function tests, pulmonary pathology reports, and other relevant data, Dr. Tuteur concluded that

> [t]here is no convincing evidence or medical support for the concept that [the Miner] was disabled as a result of his coal mine employment because of any pulmonary or respiratory health problems. Though pneumoconiosis was identified at the time of autopsy, it was not a substantially contributing cause to his premorbid disability. It did not have a material adverse effect on his respiratory or pulmonary condition. It did not have a material adverse effect on his respiratory or pulmonary function. The presence of mild simple coal workers' pneumoconiosis did in no way result in a material worsening of a totally disabling health problem either of pulmonary or other (cardiac) origin.

(DX 113) In fact, Dr. Tuteur determined that "the cardiomegaly in association with the advanced coronary artery occlusion [rather than the pneumoconiosis] clearly explains the interval development of severe disabling breathlessness…." (DX 113)

Dr. Tuteur further stated that, although the Miner did suffer from coal workers' pneumoconiosis at the time of his death, the pneumoconiosis "did not produce symptoms of physiologic impairment. Therefore, coal workers' pneumoconiosis was not a contributing cause to death by myocardial infarction. Neither the inhalation of coal mine dust nor the development of pneumoconiosis hastened his death." (DX 113) Dr. Tuteur finally concluded that, "Had [the Miner] never worked in the mining industry, his clinical course would have been no different…." (DX 113)

Dr. Robert Farney

Dr. Robert Farney examined the Miner on September 25, 1992, and prepared a medical report dated May 14, 2003. (DX 119) Dr. Farney did not cite to every source he examined before preparing his medical report, although it is clear that, at the very least, he examined notes

from his 1992 examination of the Miner and medical reports from other physicians, including Dr. Naeye, Dr. Poitras, Dr. Lincoln, and Dr. Perper. (DX 119)

After Dr. Farney met with the Miner in 1992, he concluded that

> the patient had normal pulmonary function measurements, normal arterial blood gas studies, normal physical examination [except for obesity] (i.e. no sign of pulmonary disease or impairment of airflow), and no radiographic evidence of pneumoconiosis of any kind. I pointed out that his exposure to coal dust was limited…that he had gained weight since retirement and most important, I stated, 'I would be concerned about the possibility of coronary artery disease…It is possible that the patient should have further cardiology evaluation….'"

(DX 119)

After examining the medical reports and his prior assessment of the Miner, Dr. Farney made several conclusions. (DX 119) Dr. Farney stated that "[t]he most recent valid pulmonary function and arterial blood gas measurements were obtained six years before death (1992). These two sets of studies showed no obstruction, no restriction, no diffusion impairment and normal blood gases." (DX 119) Dr. Farney concluded that the "studies obtained by Dr. Poitras in 1999 were not valid. The arterial blood gas measurements at rest were valid, but the exercise gases were venous (i.e. results of exercise blood gas measurements were not valid.) Any conclusions based upon these studies would be erroneous." (DX 119)

Dr. Farney also claimed that "[c]oal workers' pneumoconiosis is a slowly progressive process and does not usually progress after removal from exposure unless there is at least advanced simple pneumoconiosis. Therefore, whatever pulmonary impairment was present at the time of [the Miner's] death in 1999 could not have occurred secondary to pneumoconiosis." (DX 119)

Dr. Farney also stated that, although there were some autopsy findings of emphysema, "the extent of this process would not have been sufficient to cause symptoms and there was in fact no evidence of clinical impairment that could be attributed to emphysema." (DX 119)

Finally, in sum, Dr. Farney concluded that "[c]oal mine employment did not cause any pulmonary or respiratory disability and did not contribute one iota to the cause of death." (DX 119)

Dr. Joseph Tomashefski

Dr. Joseph Tomashefski prepared a medical report dated November 5, 2004. (EX 1) Before drafting his report, Dr. Tomashefski reviewed medical reports from other physicians, an autopsy report, chest x-rays, arterial blood gas studies, pulmonary function tests, the Miner's death certificate, an amended death certificate, four autopsy slides, the Miner's medical history, and a transcript of the deposition of Dr. E. A. Leis. (EX 1)

Dr. Tomashefski noted that the Miner worked as an underground coal miner for over ten years. (EX 1) Prior to working as a coal miner, he was a hard rock miner for twenty-five years. (EX 1) His work often required "drilling and blasting." (EX 1)

The Miner complained of "shortness of breath, coughing and wheezing." (EX 1) Test results suggested that the Miner had a disabling pulmonary impairment. (EX 1) Although pulmonary function tests showed "only mildly reduced FVC and FEV1, with normal FEV1/FVC ratio, MVV, TLC and DLCO," a blood gas study from 1999 showed oxygen desaturation during exercise. (EX 1)

Dr. Tomashefski noted that "[a]ccording to the amended death certificate, [the Miner] died on June 4, 1999 at the age of sixty-five years. The immediate and underlying causes of death are respectively listed as acute myocardial infarction due to occlusive coronary artery disease. Contributing causes of death are listed as cardiomegaly and coal workers' pneumoconiosis." (EX 1)

Dr. Tomashefski found that

> [b]ased on my review of the medical records, autopsy report, and slides of [the Miner's] lung tissue, it is my opinion that his sudden death was due to a massive myocardial infarct secondary to advanced coronary atherosclerosis. The intense pulmonary capillary congestion seen in his lungs is consistent with acute cardiac failure.
>
> It is also my opinion, based on a few scattered coal macules, that [the Miner] did have very mild simple coalworkers' pneumoconiosis. In my opinion, [the Miner's] simple coalworkers' pneumoconiosis is of too mild a degree to have caused him any respiratory symptoms or respiratory impairment. It is also my opinion, within reasonable medical certainty, that [the Miner's] mild simple coalworkers' pneumoconiosis is too mild to have either caused or contributed to his death. My opinion regarding the mild degree of pneumoconiosis is consistent with the predominance of radiographic data showing no evidence of pneumoconiosis, and [the Miner's] nearly normal pulmonary function test results.
>
> It is also my opinion that there is negligible fibrosis or emphysema in [the Miner's] lung tissue. Dilated airspaces without significant alveolar septal destruction as seen in [the Miner's] lung are frequently seen beneath the visceral pleura of the upper lobes or may be a consequence of aging. The histological diagnosis of emphysema requires that there be evidence of alveolar destruction in addition to airspace dilatation. [The Miner's] pulmonary function test results do not support the diagnosis of significant obstructive lung disease due to emphysema, or restrictive disease due to interstitial fibrosis.

(EX 1)

In sum, Dr. Tomashefski stated that the Miner's "progressive respiratory symptoms of dyspnea and wheezing were the result of coronary artery disease and hypertensive cardiomegaly which resulted in impaired cardiac function and cardiac failure." (EX 1) The Miner's "oxygen desaturation on exercise [was] not due to intrinsic lung disease, but [was] probably the result of cardiac dysfunction." (EX 1) Dr. Tomashefski believed that the Miner "would have died at the same time and in the same manner even if he had never worked as a coal miner." (EX 1)

Old Evidence

Dr. Michael Lincoln

Dr. Michael Lincoln examined the Miner on September 18, 1992. (DX 28) Dr. Lincoln reviewed the Miner's employment history, smoking history, and medical history. (DX 28) He also reviewed the results of a chest x-ray, a pulmonary function test and an arterial blood gas test. (DX 28)

Dr. Lincoln determined that the Miner had interstitial lung disease with chest restriction, pleural thickening and chronic bronchitis. (DX 28) Dr. Lincoln could not determine the etiology of the interstitial lung disease, although he stated that it was "probably not due to coal or hard rock mining." (DX 28) He was also unaware of the etiology of the pleural thickening, although he believed it "could be asbestos." (DX 28) He believed that the Miner's chronic bronchitis was due to dust exposure. (DX 28)

Dr. Lincoln believed that the Miner's interstitial disease caused mild to moderate impairment. (DX 28) He further stated that the Miner's pleural thickening generally caused no disability and that chronic bronchitis "without obstruction is non-disabling." (DX 28) Finally, Dr. Lincoln concluded that twenty to fifty percent of the Miner's impairment could be attributed to his lung disease and that fifty to eighty percent of the Miner's impairment could be attributed to his back and knee pain. (DX 28)

Dr. Robert Farney

Dr. Robert Farney examined the Miner on September 25, 1992. (EX 1) The purpose of this examination "was to perform an independent medical examination with respect to coal workers' pneumoconiosis and respiratory impairment." (EX 1) Dr. Farney reviewed the Miner's medical history, the Miner's employment history, chest x-rays, pulmonary function tests, arterial blood gas tests, and routine laboratory data. (EX 1)

Dr. Farney noted that "[o]ver the last several years, [the Miner] has continued to have recurrent difficulties with shortness of breath, coughing, and wheezing. His cough is considered minimal….He estimates sputum production to be about one teaspoon per day, which is mixed with blood." (EX 1)

After examining the Miner, Dr. Farney determined that "[t]he patient presents an obese, otherwise healthy appearing Caucasian male." (EX 1) After performing several tests, Dr. Farney found that the arterial blood gas measurements were within normal limits and that the

pulmonary function measurements "demonstrated slight chest restriction, but no evidence of obstructive airways disease. The flow volume loops were within normal limits." (EX 1)

Dr. Farney concluded that "[t]he patient's clinical history suggests the possibility of chronic obstructive airways disease or asthma, however, the present data does not substantiate that diagnosis." (EX 1) Dr. Farney further stated that the "constellation of data in this case does not support the diagnosis of coal workers' pneumoconiosis," and that, although the Miner was noted to have mild chest restriction and abnormalities of the pleura, and although the exact etiology of the pleural fibrosis was unclear, "there is no relationship of the pleural abnormality and coal dust exposure." (EX 1) Dr. Farney stated that he was concerned that the Miner might have coronary artery disease. (EX 1) He also suspected "that some symptoms of effort intolerance could be related to obesity." (EX 1)

Dr. Jean-Maurice Poitras

Dr. Jean-Maurice Poitras examined the Miner on March 30, 1999. (DX 7) He sent the Miner to have a chest x-ray, a pulmonary function test, and an arterial blood gas test performed. (DX 7) Dr. Poitras also reviewed the Miner's employment history, smoking history, and medical history before drafting his medical report.

During the examination, the Miner complained of slight chronic wheezing, dyspnea and orthopnea. (DX 7) The pulmonary function study, performed on April 6, 1999, showed severe restriction and obstruction. (DX 7) Values recorded during the arterial blood gas study, performed on April 7, 1999, noted oxygen desaturation. (DX 9) At rest, the Miner's P02 was 70.1 and the PC02 was 37.5. (DX 9) However, upon exercise, the Miner's P02 was 45.8 and the PC02 was 35.2. (DX 9)

Dr. Poitras determined that the Miner had severe obstructive and restrictive lung disease and "a fib. structural heart disease, cardiomegaly from pulmonary hypertension." (DX 7) Dr. Poitras determined that these cardiopulmonary problems were caused by exposure to coal dust, exposure to uranium, and the Miner's smoking history, which consisted of approximately one pack per day for twenty years. (DX 7) Dr. Poitras further determined that the Miner was impaired due to the severe obstructive and restrictive disease and due to the Miner's "significant reduction in diffusing capacity." Dr. Poitras noted that the Miner's restrictive disease could have been caused by coal dust, "but [was] probably due more to uranium exposure." (DX 7) He found that the obstructive disease was due to smoking and his work around coal dust and uranium. (DX 7)

Dr. Edward Leis

Dr. Edward Leis was the Deputy Chief Medical Examiner who performed the Miner's autopsy on June 5, 1999. (DX 5) Dr. Leis determined that the Miner had occlusive coronary artery disease, which involved an eighty percent occlusion of the right coronary artery, an eighty percent occlusion in the left anterior descending coronary artery and an acute thrombotic occlusion of the left circumflex artery. (DX 5) Dr. Leis further found that the Miner had cardiomegaly with biventricular dilation and pulmonary emphysema, anthracosis, and mild

arterial changes. (DX 5) In sum, Dr. Leis opined that the Miner "died of an acute myocardial infarction, secondary to thrombotic occlusion of the left circumflex artery. Significant blockage of all three main coronary arteries was identified. The pulmonary changes [such as thickening of the intima of small pulmonary arteries] are consistent with coal workers' pneumoconiosis." (DX 5)

Dr. Leis was deposed on January 27, 2000. (DX 49) First, Dr. Leis confirmed that no other medical records regarding the Miner were reviewed prior to performing the autopsy report. (DX 49, pgs. 4-5)

During this deposition, Dr. Leis noted that two death certificates were issued in this case. (DX 49, pgs. 5-6) The first certificate was issued on June 8, 1999. (DX 49, pg. 5) On this certificate, Dr. Leis noted that the final cause of death would be determined after the toxicology results were released. (DX 49, pg. 5) The final certificate was issued on July 14, 1999. (DX 49, pg. 6) Dr. Leis assigned "acute myocardial infarction due to occlusive coronary artery disease" as the immediate cause of death. (DX 49, pg. 6) Dr. Leis also listed other causes of death. He stated that, "[u]nder 'Acute Myocardial Infarction' that's where I listed 'Occlusive Coronary Artery Disease.' And then in Part II, under 'Other Significant Conditions,' I listed cardiomegaly and coal workers' pneumoconiosis." (DX 49, pg. 6)

When asked how he determined that the Miner's coal workers' pneumoconiosis was a significant condition that contributed to the Miner's death, Dr. Leis stated that

> we had a history that he was a … coal miner in years past. And then the rest was based upon the gross and microscopic examination of lung tissue….Grossly, the lungs – the surfaces of the lungs were almost completely black and the lungs had an increased firmness throughout. Microscopically, the findings were consistent with coal worker's pneumoconiosis in that there was black anthracotic pigment [of less than three millimeters in size] trapped in tissue which was focally scarred, and there were some associated changes of emphysema.

(DX 49, pg. 7) Moreover, when asked whether the Miner's simple coal workers' pneumoconiosis caused his death, Dr. Leis replied that "[b]ecause of the way I put it on the death certificate, it would have contributed to it." (DX 49, pg. 8) He went on to state that the Miner's coal workers' pneumoconiosis contributed to the Miner's death by causing "some impairment in [the Miner's] ability to oxygenate his blood, [and] increased his susceptibility to heart damage associated with his coronary artery disease." (DX 49, pgs. 8-9) In sum, Dr. Leis stated that the Miner's coal workers' pneumoconiosis contributed to some pulmonary problems, which would have had an effect on his heart condition. (DX 49, pg. 9)

However, although Dr. Leis found that the Miner's pneumoconiosis contributed to his death, he also found that, although the Miner had coal workers' pneumoconiosis, he still would have died of the acute myocardial infarction due to occlusive coronary artery disease. (DX 49, pg. 8) Moreover, Dr. Leis stated that the Miner would have died at the same time even in the absence of coal workers' pneumoconiosis. (DX 49, pg. 9)

Dr. Joshua Perper

Dr. Joshua Perper prepared a medical report dated November 1, 1999. (DX 33) When drafting his report, Dr. Perper relied on the Miner's employment records, the autopsy report, four autopsy slides, chest x-rays, arterial blood gas studies and pulmonary function tests. (DX 33)

Dr. Perper noted that the Miner had slight chronic wheezing, shortness of breath upon minimal exertion, and occasional shortness of breath while sleeping with orthopnea. (DX 33) Dr. Perper also noted the increase in the anterior-posterior diameter of the chest, which he believed was consistent with pulmonary emphysema. (DX 33) Dr. Perper also noted that the results of the arterial blood gas test performed on April 7, 1999 showed oxygen desaturation. (DX 33) Based, at least in part, on the arterial blood gas study, Dr. Perper found obstructive and restrictive lung disease with hypoxemia. (DX 33)

Dr. Perper also reviewed the autopsy slides. (DX 33) After reviewing the slides, he determined that the Miner had "simple coal workers' pneumoconiosis, macular and micro-nodular, slight to moderate, centrilobular emphysema, slight to moderate, [and] sclerosis of intra-pulmonary blood vessels indicative of pulmonary hypertension." (DX 33)

Based on all of the information he reviewed, Dr. Perper concluded that the Miner "had severe objectively demonstrated obstructive and restrictive lung disease and hypoxemia, due to significant simple mine workers' pneumoconiosis, and associated emphysema, interstitial fibrosis and sclerosis of pulmonary vessels, consistent with pulmonary hypertension and cor pulmonale." (DX 33) Dr. Perper related the Miner's pneumoconiosis to his occupational exposure to rock dusts, including coal dust. (DX 33)

Dr. Perper also concluded that the Miner's "pneumoconiosis was a substantial contributory cause of his death through severe pulmonary dysfunction and hypoxemia that caused, precipitated or aggravated a final incident of myocardial infarction that occurred on the background of severe arteriosclerotic heart disease and related coronary thrombosis and myocardial infarction." (DX 33) Dr. Perper believed that the Miner's coal workers' pneumoconiosis was a substantial contributory cause of [the Miner's] death "based on the severe objectively demonstrated obstructive and restrictive pulmonary disease, evidenced in the clinical testing and the findings of pneumoconiosis and associated findings of chronic emphysema, interstitial fibrosis and pulmonary sclerosis consistent with pulmonary hypertension and cor pulmonale, that were demonstrated at the autopsy." (DX 33)

Dr. Joshua Perper-Supplemental Report

On October 2, 2000, after reviewing his former report, Dr. Richard Naeye's report, Dr. Raphael Caffrey's report, Dr. Leis's deposition, and the chest x-ray reports of Drs. William Scott and Paul S. Wheeler, Dr. Perper submitted an updated medical report. (CX 1) In this supplemental report, Dr. Perper stated that

> pneumoconiosis does not have an effect on speeding up the development of the miner's cardiovascular disease…. [However,] speeding the development of

> cardiovascular disease is not the only mechanism by which pneumoconiosis can hasten a patient [*sic*] death. As a matter of fact the increased susceptibility to heart damage by the coal workers' pneumoconiosis related respiratory impairment (as stated by the prosector) is a reasonable and effective hastening mechanism of death.

(CX 1) Dr. Perper did not believe "that [the Miner] would have died at the same time as he did, if he would not have had coal worker's pneumoconiosis." He found that Dr. Leis's statement to the contrary "stands in contradiction with his determination that 'some impairment in [the Miner's] ability to oxygenate blood, increased his susceptibility to heart damage associated with his damage associated with his coronary artery disease.' Clearly an 'increased susceptibility to heart damage' can hasten or precipitate a death." (CX 1)

In sum, even after examining Dr. Naeye's report, Dr. Caffrey's report, Dr. Leis's deposition, and the chest x-ray reports of Dr. Scott and Dr. Wheeler, Dr. Perper maintained that the Miner's total disability was due to pneumoconiosis and that his death was due to pneumoconiosis. (CX 1)

Dr. Raphael Caffrey

On February 15, 2000, Dr. Caffrey prepared a medical report. (DX 45) Before preparing his report, Dr. Caffrey examined chest x-rays, arterial blood gas tests, ECG tracings, the Miner's death certificate, the autopsy, four autopsy slides, and other physicians' medical reports. (DX 45) However, some of the information he reviewed was incomplete. He noted that Dr. Perper's report contained the only work history, smoking history, and medical history that he received. (DX 45) He also indicated that he only received two of three pages of Dr. Leis's autopsy report. (DX 45)

Dr. Caffrey stated that, based on the information he reviewed, the Miner

> did have a mild degree of simple coal workers' pneumoconiosis. The simple coal workers' pneumoconiosis, in my opinion, did not contribute to, cause, or hasten his death. [The Miner] died of coronary artery thrombosis with secondary acute myocardial infarction. He had a heart, which weighed 750 gms which was over twice normal. There is definitely no cause and effect relationship between coronary artery atherosclerosis, myocardial infarction, and simple coal workers' pneumoconiosis. There is no association between working in a coal mine or uranium mine and the development of coronary artery atherosclerosis and/or myocardial infarction.

(DX 45)

Thus, Dr. Caffrey disagreed with the opinions of Drs. Perper and Poitras, who stated that the Miner's coal workers' pneumoconiosis caused, contributed to or hastened death. (DX 45)

Dr. Caffrey also seemed to disagree with Dr. Poitras's finding that the patient was disabled by pulmonary disease, and he certainly disagreed with Dr. Poitras's determination that coal workers' pneumoconiosis caused this disability. (DX 45) He stated that "if, indeed, [the Miner] had this significant pulmonary problem, in my opinion, it was definitely not due to the amount of simple coal worker's pneumoconiosis he had." (DX 45) Rather, Dr. Caffrey believed that the Miner's twenty-year smoking history or pulmonary congestion or edema secondary to his heart disease were likely to blame for any disabling pulmonary problems. (DX 45)

In sum, Dr. Caffrey determined that the Miner "did have a mild degree of simple coal workers' pneumoconiosis…and he had centrilobular emphysema of a mild degree."[3] Dr. Caffrey also determined that, "It is my opinion though, that the mild degree of simple coal workers' pneumoconiosis did not contribute to, hasten, or cause his death." (DX 45)

Dr. Richard Naeye

Dr. Richard Naeye drafted a medical report on March 7, 2000. (DX 49) Before preparing his report, Dr. Naeye reviewed the Miner's autopsy report, the death certificate, the Miner's employment history, smoking history and medical history, chest x-rays, pulmonary function studies, arterial blood gas tests, and four autopsy slides. (DX 49)

Dr. Naeye noted that in 1999, "eight years after he had quit mining coal, atrial fibrillation, wheezing and dyspnea on minimal exertion and occasional orthopnea appeared." (DX 49) After reviewing the Miner's test results, Dr. Naeye noted that a 1993 pulmonary function study produced an abnormal FEF 25-75 value and that arterial blood tests from 1993 and 1999 showed "severe arterial oxygen desaturation" during exercise. (DX 49) After reviewing the autopsy report, Dr. Naeye noted that

> both ventricles of [the Miner's] heart were dialted and a large, recent infarction was found in the posterior wall of his left ventricle. Both his right coronary artery and the left anterior descending branch of his left coronary artery were 80% occluded by arteriosclerosis. A fresh thrombus occluded the circumflex branch of his left coronary artery. It was no doubt responsible for the large myocardial infarction and death.

(DX 49)

After summarizing all of the information, Dr. Naeye concluded that the Miner did indeed suffer from simple coal workers' pneumoconiosis. (DX 49) However, although the physician determined that the minimal findings required to make the diagnosis of simple coal workers' pneumoconiosis were present, including "a moderate number of anthracotic macules, occasionally with a small amount of associated fibrous tissue and rare tiny birefringent crystals" and rare focal emphysema, "which constitutes [less than] 1% of the total emphysema in the lung

[3] At one point in his opinion, Dr. Caffrey opined that the Miner's emphysema was "mild" and was caused by his cigarette smoking rather than coal dust exposure. However, Dr. Caffrey later stated, "I cannot prove whether the centrilobular emphysema was due to his cigarette smoking, which I believe it was, with possibly a minor contribution from the coal worker's pneumoconiosis." (DX 45)

tissues available for review," he found that the coal workers' pneumoconiosis was "far too mild to have: (a) produced any measurable effects on lung function, (b) caused any disability, (c) prevented him from mining coal, (d) had any role in hastening or causing his death." (DX 49) Instead, Dr. Naeye believed that the Miner's atrial fibrillation, wheezing, dyspnea on minimal exertion, occasional orthopnea, arterial oxygen desatruation at rest and with exercise, and death were due to "[m]yocardiac malfunction due to severe coronary arteriosclerosis, damage to the microcirculation of his heart by cigarette smoking and at the end a coronary artery thrombosis." (DX 49)

Dr. Richard Naeye – Supplemental Report

On October 7, 2000, Dr. Naeye submitted a supplemental report. (DX 60) In this report, Dr. Naeye stated that

> [t]here was no doubt that [the Miner] had respiratory signs and symptoms. As will be seen they were secondary to cardiac damage and failure. He had a history of substantial damage to his heart by ateriosclerosis in large arteries and damage to small arteries and arterioles by his heavy cigarette smoking. They in turn led to atrial fibrillation which led to a reduction in cardiac output and a resulting evidences myocardial insufficiency and heart failure. The result was wheezing, dyspnea on exertion and arterial blood oxygen desaturation which increased with exercise. Such heart failure fluctuates and that explains why [the Miner's] arterial blood PO2s fluctuated from time to time. Such heart failure also increases pulmonary venous pressure with resulting increases in pulmonary arterial blood pressure and produces cor pulmonale. There was no primary disease, including [coal workers' pneumoconiosis] in the lungs of [the Miner] that could have caused chronic cor pulmonale. It was all secondary to his chronic left sided heart failure.

(DX 60)

Dr. Naeye also stated that, "taken together all of the diseases in [the Miner's] lungs were far too mild to have had any significant effect on lung function. These include (a) the very tiny coal dust macules lesions in his lungs, most of which have no associated fibrosis or focal emphysema, (b) the two types of emphysema." (DX 60)

Dr. Richard Naeye – Supplemental Report

On October 17, 2000, Dr. Naye provided another supplemental report. (DX 72) In this report, he reiterated his belief that the Miner's coal workers' pneumoconiosis was "too mild to cause any abnormalities in lung function, contribute to disability or hasten death." (DX 72) He stated that the Miner's death was caused by his smoking history and severe artiosclerosis in his major coronary arteries. (DX 72)

Dr. Naeye also stated that he disagreed with many of Dr. Leon Cander's assertions because

> Dr. Cander does not understand the major problem that consultant pathologists face in reviewing the lung tissues of coal miners and how this problem affects their reports. Unfortunately, prosectors most often take tissues for microscopic examination from the blackest, most damaged areas of the lungs they can find at autopsy. This leads consultant pathologists to very often judge [coal workers' pneumoconiosis] to be severe when overall it was mild or moderate in severity. The [coal workers' pneumoconiosis] in lung tissues taken at autopsy from [the Miner] is mild. This is confirmed by the inability of radiologists to identify the disorder on x-rays. The fact that it was mild is further confirmed by the results of the 1993 pulmonary function studies which produced normal FEV1, FVC and FEF max values. This was two years after he quit mining coal. Simple CWP does not progress after a worker leaves the exposure to coal mine dust. This is important because later arising abnormalities in lung function sometimes occur in coal miners that are the consequence of cigarette smoking and heart failure.

(DX 72) Thus, Dr. Naeye found that "Dr. Cander is incorrect in his judgment that [coal workers' pneumoconiosis] had any role in the disability and death of [the Miner]. The [coal workers] pneumoconiosis] was far too mild to have had such a role." (DX 72)

Dr. Leon Cander

Dr. Leon Cander prepared a consultation report for this case on July 13, 2000. (DX 59) Although Dr. Cander did not provide a list detailing exactly what materials he reviewed, it is clear that he examined the Miner's employment history, smoking history and medical history, chest x-rays, pulmonary function tests, arterial blood gas tests, an electrocardiogram, other physicians' medical reports, and the Miner's autopsy report. (DX 59)

After examining arterial blood gas studies dated September 18, 1992 and April 7, 1999, Dr. Cander noted that the studies demonstrated a "marked decrease in PO2 during exercise which documents the presence of chronic, disabling lung disease in a man who has a proven history of almost 15 years of coal mine employment." (DX 59)

Dr. Cander also determined that the evidence he reviewed showed that the Miner suffered from severe pulmonary problems. (DX 59) Dr. Cander stated that Dr. Leis' autopsy findings "indicated thickening of both the left and right ventricular walls as well as thickening of the lining of the small pulmonary arterioles. The thickness of the right ventricular wall and the changes in the pulmonary arterioles document the presence of chronic elevation of the pulmonary artery pressure and resultant cor pulmonale." (DX 59) The arterial blood gas studies "demonstrated that hypoxia caused by chronic lung disease was the cause of the pulmonary hypertension and cor pulmonale." (DX 59) Dr. Cander stated that the "presence of hypoxemia and pulmonary hypertension represent serious and independent risk factors for the sudden spontaneous development of life-threatening cardiac rhythm disturbances. (DX 59) Based on the information available to him, Dr. Cander determined that the Miner's death could be attributed to cardiac arrhythmia, which was at least partly related to the Miner's hypoxemia and pulmonary hypertension. (DX 59)

In sum, Dr. Cander concluded that the pathologic evidence proved that the Miner suffered from pneumoconiosis. (DX 59) He further found that "[t]he coal workers' pneumoconiosis caused hypoxemia severe enough to meet the disability standard of the Federal Black Lung Program, documenting the presence of disabling pneumoconiosis." (DX 59) Finally, he determined that

> [a]lthough [the Miner's] immediate cause of death was listed as occlusive coronary artery disease causing an acute myocardial infarction, in fact the immediate cause of death was a fatal cardiac arrhythmia which was causally related, at least in part, to the hypoxemia and pulmonary hypertension caused by the coal workers' pneumoconiosis. Thus, [the Miner's] pneumoconiosis contributed significantly as a cause of his death and hastened it.

(DX 59)

Dr. Cander stated that he disagreed with Dr. Naeye's opinion that Miner's coal workers' pneumoconiosis was "too mild" to cause any disability. (DX 59)

Dr. Gregory J. Fino

Dr. Gregory Fino prepared a report for this case dated July 24, 2000. (EX 3) Dr. Fino stated that he had "reviewed all of the medical records that you have been able to develop regarding [this Miner]." (EX 3) Although he did not specify exactly what sources had been reviewed, it is clear that the Miner's employment records, medical reports from other physicians, chest x-ray reports, arterial blood gas studies, pulmonary function tests, the Miner's death certificate, the autopsy report, a transcript of the deposition of Dr. Leis, and pathology reports were examined. (EX 3)

Dr. Fino noted that the Miner had previously been employed for fourteen years as an underground coal miner. (EX 3) He was last employed in 1991 or 1992. (EX 3) It was also noted that, in 1992, the Miner "complained of shortness of breath, wheezing, coughing, and sputum production." (EX 3)

After examining the Miner's pulmonary function and arterial blood gas test results, Dr. Fino found that a 1992 pulmonary function test showed "no ventilatory impairment due to any obstructive or restrictive ventilatory defect." (EX 3) Other pulmonary function studies were performed, including on September 25, 1992 and April 6, 1999, but these studies were invalid due to a failure to include the tracings with the 1992 report and a poor effort noted on the 1999 tracings. (EX 3) A 1992 arterial blood gas test showed "mild resting hypoxia that did worsen with exercise." (EX 3) Moreover, the results of a 1999 arterial blood gas test showed "a drop in the PO_2 with exercise." (EX 3)

After examining the death certificate and the autopsy report, Dr. Fino noted that "[t]he immediate cause of death was listed as acute myocardial infarction due to occlusive coronary

artery disease." (EX 3) He further noted that the prosector "felt that the changes in the lungs were consistent with coal workers' pneumoconiosis." (EX 3)

Dr. Fino determined that the "diagnosis of a coal mine dust-related lung disease cannot be established merely by a history of respiratory complaints and physical examination….Objective tests are absolutely essential in distinguishing pneumoconiosis from non-occupational pulmonary disorders." (EX 3) After reviewing the Miner's test results, Dr. Fino noted that, "What I see in this case is someone who, on the only acceptable pulmonary function study from 1992, had normal ventilatory function. The MVV was normal." (EX 3) Although the arterial blood gas tests showed significant oxygen desaturation with exercise, which was disabling, Dr. Fino found that

> [o]xygen desaturation can occur as a result of interstitial lung disease or occupational pneumoconiosis. However, the amount of pneumoconiosis that was present on the pathology report was far too mild to have accounted for this marked drop in the blood oxygen level. Therefore, I do not feel comfortable attributing this man's oxygen destaturation to interstitial lung disease, pulmonary fibrosis or even pneumoconiosis.

(EX 3) Thus, Dr. Fino did not believe that the Miner's "disability, which was present prior to his death, [could] be attributed to the inhalation of coal mine dust."[4] (EX 3) Dr. Fino based this opinion in part on the following reasoning: "If this man had such severe lung disease due to either smoking or coal mine dust, one would think that there would be some attempt at treatment of his lung condition with medications. However, as of 3/30/99, there was no evidence of treatment with bronchodialators or, for that matter, any type of lung treatment." (EX 3)

In sum, Dr. Fino stated that

> I think the records clearly show that this man died of an acute myocardial infarction. There is no reason to suggest that any type of lung disease contributed to this death. There is no causal association between coal mine dust inhalation, or for that matter chronic lung disease if present, and death from coronary artery disease.

(EX 3)

Discussion

In the November 6, 2001 Decision and Order, Administrative Law Judge Paul H. Teitler determined that the Miner was totally disabled due to pneumoconiosis. The Employer then requested a modification on the basis of a mistake in a determination of fact. It submitted medical reports from Dr. Farney, Dr. Tuteur and Dr. Tomashefski to support this allegation. Since the Employer requested the modification, the burden of proof is placed upon the Employer

[4] However, he was unable to pinpoint exactly what did cause the oxygen desaturation during exercise. (EX 3) Congestive heart failure and centriacinar emphysema were ruled out as causes of the drop in blood oxygen level with exercise since they were not, in his opinion, severe enough to have an effect on the blood oxygen level. (EX 3)

to prove that the prior decision awarding benefits contained a mistake of fact. See Wojtowicz v. Duquesne Light Co., 12 B.L.R. 1-162, 1-164 (1989).

As is noted above, the finder of fact has broad discretion to correct mistakes of fact. O'Keeffe v. Aerojet-General Shipyards, 404 U.S. 254, 256 (1971). A mistake of fact may be demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." Id. The undersigned must thus assess, de novo, all of the evidence to determine if an error was made in the determination that the Miner was totally disabled due to pneumoconiosis and that his death was hastened by pneumoconiosis. See Branham v. Bethenergy Mines, Inc., 20 B.L.R. 1-27, 1-31 (1996).

Was There a Mistake of Fact in the Living Miner's Claim?

Generally, in order to establish entitlement in a living miner's claim, it must be established that the miner was totally disabled due to pneumoconiosis at the time of death. See 20 C.F.R. § 718.204(a)(2003).[5] The Tenth Circuit, where this case originated, has determined that a miner shall be considered totally disabled due to pneumoconiosis if pneumoconiosis is "at least a contributing cause" of the Miner's disability. Mangus v. Director, OWCP, 882 F.2d 1527, 1531-1532 (10th Cir. 1989).[6] The Tenth Circuit further held that

> the claimant bears the burden of proving that his total disability is due to pneumoconiosis by a preponderance of the evidence. Baumgartner v. Director, Office of Workers' Compensation Programs, 9 Black Lung Rep. (MB) 1-65, 1-66 (1986); Gee v. W.G. Moore and Sons, 9 Black Lung Rep. (MB) 1-4,1-6 (1986). We believe this burden would be inappropriately heavy were we to require that he or she also prove that the causal nexus fit a description which is embodied in such nebulous terms as 'significant' or 'substantial.'

Id. at 1531.

First, as is noted above, it does not appear that the Employer contests that the Miner was totally disabled at death. In its brief, the Employer stated, "In order for disability causation to be established, … pneumoconiosis must be more than a de minimis or speculative cause of his disability prior to his death….Where, as here, the credible evidence established that the impairment is due to conditions other than pneumoconiosis, such as heart disease, entitlement is precluded." Moreover, none of the new medical reports submitted by the Employer alleged that the Miner was not totally disabled. In fact, Dr. Tuteur clearly stated that the Miner was totally

[5] Given that this claim was filed after March 31, 1980, the date that Part 718 was implemented, the claim must be adjudicated under those regulations. Revised regulations became effective on January 19, 2001, and are applicable to claims pending on that date. The Decision and Order in this case was not issued until November 6, 2001. However, it must be noted that the revised regulations do not apply to certain regulations, including those at 20 C.F.R. §§ 725.309, 725.310, 425.414, 725.492, 725.493, 725.494 and 725.495.

[6] See also Pittsburgh & Midway Coal Mining Co. v. Sanchez, 2001 WL 997947, Case No.: 00-9538, pg. *724 (10th Cir. Aug. 31, 2001) (declining to apply the causation standard set forth in the amended regulations at 20 C.F.R. Sect. 708.204(c)(1) (2001), and explaining in a footnote "we apply the Mangus causation standard that was in effect when Sanchez filed for benefits in 1988." Id.).

disabled prior to his death. Thus, the only issue to examine is whether the Miner's total disability was due to pneumoconiosis.[7]

Dr. Lincoln and Dr. Farney examined the Miner in 1992. (DX 28, EX 1) Dr. Lincoln found that the Miner had interstitial lung disease with chest restriction. Although Dr. Lincoln was unable to pinpoint the etiology of the interstitial lung disease, he did not think that it was caused by coal dust exposure. (DX 28) Dr. Farney found no evidence of pneumoconiosis. (EX 1) Obviously, therefore, Dr. Farney did not conclude that the Miner was totally disabled due to pneumoconiosis.

However, the Board has determined that, when "weighing medical reports, the administrative law judge may find a doctor's opinion based on limited clinical data of less weight than conflicting reports based on more comprehensive documentation." Sabett v. Director, OWCP, 7 B.L.R. 1-299, 1-301, n. 1 (1984). The Miner died in 1999. These reports by Dr. Lincoln and Dr. Farney fail to account for any changes in the Miner's condition that occurred, or any new evidence that was acquired, in the seven years between the making of the reports and the Miner's death. The undersigned finds that these reports cannot be given the same weight as later reports that considered more evidence, including a 1999 arterial blood gas test that produced values that qualified as totally disabling.

Dr. Poitras examined the Miner in 1999, shortly before his death. (DX 7) The undersigned finds Dr. Poitras' opinion, which was based on physical examination, chest x-rays, a pulmonary function test and an arterial blood gas test, to be well-documented.[8]

Dr. Poitras determined that the Miner had severe obstructive and restrictive lung disease, and that the obstructive disease was due to smoking and his work around coal dust and uranium. (DX 7) Dr. Poitras thus found that the Miner's pneumoconiosis was "at least a contributing cause" of the Miner's disabling lung disease.

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[7] Even if the Employer would have argued that the Miner was not totally disabled, the undersigned would have found the Miner to be totally disabled. The Tenth Circuit stated in Pittsburgh & Midway Coal Mining Co. v. Sanchez that "[t]otal disability means that the impairment prevents the claimant from performing his usual coal mine work and from engaging in other comparable and gainful work available in the immediate area of his residence." 2001 WL 997947, Case No.: 00-9538, pg. *724 (10th Cir. Aug. 31, 2001).

The values obtained from a 1999 arterial blood gas study support a finding of total disability. The weight of the medical reports also supports a finding of total disability. Drs. Poitras, who examined the Miner in 1999, determined, based at least in part upon physical examination and arterial blood gas test results, that the Miner was totally disabled. Drs. Perper and Cander also found the Miner to be totally disabled. These opinions are supported by the opinion of Dr. Tuteur. Moreover, neither Dr. Farney nor Dr. Tomashefski argued that the Miner was not totally disabled. They simply stated that any disability was not due to pneumoconiosis.

Given the evidence available in this case, the undersigned finds that, prior to his death, the Miner's pulmonary impairment was so severe that he could not perform the duties of his last regular job, which included drilling and blasting rock. Thus, even if the Employer would have argued that the Miner was not totally disabled, it would not have met its burden in establishing that the Miner was not totally disabled prior to death.

[8] A documented opinion is one that sets forth the clinical findings, observations, facts and other data that the physician relied upon when making his diagnosis. Fields v. Island Creek Coal Co., 10 B.L.R. 1-19, 1-22 (1987).

The undersigned concludes that Dr. Poitras's findings are well-reasoned.[9] It is noted that since Dr. Poitras's report was written prior to the Miner's death, he did not consider the Miner's autopsy report when writing his report. However, Dr. Poitras's findings are well-reasoned even without his review of the autopsy report. The autopsy report did not discuss whether the Miner was totally disabled or whether the Miner's total disability was due to pneumoconiosis. Moreover, Dr. Poitras relied on a significant amount of data, including his impressions after personally examining the Miner, when making his determination on whether the Miner was totally disabled due to pneumoconiosis.

Dr. Leis performed the Miner's autopsy on June 5, 1999. (DX 5) However, as is noted above, the autopsy report did not discuss whether the Miner was totally disabled or whether the Miner's total disability was due to pneumoconiosis.

Dr. Perper prepared his report on November 1, 1999. The undersigned finds that Dr. Perper's report, which was made after examining employment records, the autopsy report, autopsy slides, and numerous test results, is well-documented.

Dr. Perper concluded that the Miner's severe restrictive and obstructive pulmonary disease was due to pneumoconiosis and associated centrilobular emphysema. (CX 1)

The undersigned finds Dr. Perper's report to be well-reasoned. The Employer argued in its Brief on Remand that Dr. Perper's opinion was not well-reasoned because he failed to adequately address the effect of the Miner's blocked arteries, or the effect of the Miner's twenty year smoking history, on the Miner's disability. However, the undersigned disagrees. Dr. Perper noted in his report that the Miner had "a smoking history of less than a pack of cigarettes a day between 1950 and 1979, when he stopped smoking." (DX 33) Thus, it is clear that Dr. Perper was aware of the Miner's smoking history. There is no evidence that Dr. Perper failed to adequately consider the effect that the Miner's smoking played in his total disability. In fact, the Employer submitted new evidence that seems to support Dr. Perper's finding that the Miner's severe pulmonary problems were not likely caused by cigarette smoke. Dr. Tuteur stated in his medical report that "[a]t worst, this tobacco smoke exposure puts [the Miner] at only slight increased risk for the development of tobacco smoke associated health problems…." (DX 113) Moreover, there is no evidence that Dr. Perper failed to adequately consider the effects of the Miner's blocked arteries. Dr. Perper reviewed the Miner's autopsy, which included information on the Miner's blocked arteries, when writing his report. (DX 33) He noted that the Miner's death was caused by myocardial infarction that occurred on the background of severe arteriosclerotic heart disease. However, he also determined that pneumoconiosis aggravated this myocardial infarction and that pneumoconiosis contributed to the Miner's severe restrictive and obstructive pulmonary disease.

Dr. Caffrey prepared a medical report on February 15, 2000. Dr. Caffrey stated that,

> [i]n Dr. Perper's report on page 3 he notes that Dr. Poitras concluded that the patient was markedly impaired because of severe obstructive and restrictive

[9] A reasoned opinion is one where the underlying documentation and data are found to be adequate to support the physician's conclusions. Fields v. Island Creek Coal Co., 10 B.L.R. 1-19, 1-22 (1987).

> disease, pulmonary fibrosis and significant reduction and diffusing capacity. If, indeed, [the Miner] had this significant pulmonary problem, in my opinion, it was definitely not due to the amount of simple coal worker's pneumoconiosis he had. Certainly, the patient has a significant smoking history of 20 years plus. It is quite likely that he may have had pulmonary congestion or edema secondary to his heart disease, which was caused by his severe atherosclerosis of his coronary arteries and not related to his simple coal worker's pneumoconiosis. The centrilobular emphysema, which [the Miner] had, in my opinion, was mild and most of it certainly was due to his cigarette smoking.

(DX 45)

The undersigned does not find Dr. Caffrey's report to be well-reasoned. First, Dr. Caffrey relied upon the Miner's autopsy report when drafting his medical report, however he only received two of three pages of the autopsy report. (DX 45) Dr. Caffrey's report also contained inconsistencies, such as when Dr. Caffrey stated once that most of the Miner's pulmonary emphysema "certainly was due to cigarette smoking," but later stated, "I cannot prove whether the centrilobular emphysema was due to cigarette smoking…." (DX 45)

Dr. Naeye submitted a report on March 7, 2000. (DX 49) The undersigned finds Dr. Naeye's report, which was based upon the Miner's autopsy report, the Miner's employment history, smoking history and medical history, and numerous test results, to be well-documented.

In this report, Dr. Naeye clearly explained his findings. He determined that, although the Miner had coal workers' pneumoconiosis, it was much too mild to have caused any disability. (DX 49) Instead, Dr. Naeye determined that the miner's wheezing, dyspnea and arterial oxygen desaturation were due to a variety of causes, including myocardiac malfunction due to severe coronary arteriosclerosis, damage to the microcirculation of his heart by cigarette smoking and a coronary artery thrombosis. (DX 49) Dr. Naeye also provided a supplemental opinion dated October 17, 2000. (DX 72) In this opinion, Dr. Naeye stated, "[s]imple CWP <u>does not</u> progress after a worker leaves the exposure to coal mine dust. This is important because later arising abnormalities in lung function sometimes occur in coal miners that are the consequence of cigarette smoking and heart failure." (emphasis added) (DX 72)

The undersigned finds that this statement is contrary to the Act. The undersigned finds that coal workers' pneumoconiosis can progress after a worker leaves exposure to coal mine dust. Dr. Naeye seems to have based his determination that the Miner's totally disabling lung function was due to heart problems and cigarette smoking, rather than due to pneumoconiosis, on the faulty determination that the Miner's total disability could not be due to pneumoconiosis since pneumoconiosis could not progress in the years following his retirement. The undersigned must therefore give Dr. Naeye's conclusion that the Miner's totally disabling lung function was due to heart problems and cigarette smoking, rather than due to pneumoconiosis, diminished probative weight.

Dr. Cander prepared a report on July 13, 2000. (DX 59) The undersigned finds Dr. Cander's report, which was based upon multiple test results, the Miner's autopsy report, other

physicians' medical reports, and the Miner's employment, smoking and medical histories, to be well-documented.

Dr. Cander determined that the Miner's total disability was due to pneumoconiosis. (DX 59) He specifically noted his disagreement with Dr. Naeye's findings that the Miner's coal workers' pneumoconiosis was too mild to cause any disability. (DX 59)

The undersigned also finds Dr. Cander's report to be well-reasoned. His opinions were clearly stated and explained. (DX 59)

Dr. Fino submitted a report on July 24, 2000. (EX 3) The undersigned finds Dr. Fino's report, which was based upon the Miner's autopsy report, the Miner's employment history, the deposition of Dr. Leis, and numerous test results, to be well-documented.

Dr. Fino stated in his report that he did not believe that the Miner's disability was due to the inhalation of coal mine dust. (EX 3) He based this determination on his reasoning that, "If this man had such severe lung disease due to either smoking or coal mine dust, one would think that there would be some attempt at treatment of his lung condition with medications. However, as of 3/30/99, there was no evidence of treatment with bronchodialators or, for that matter, any type of lung treatment." (EX 3)

The undersigned does not find Dr. Fino's opinion to be well-reasoned. The undersigned disagrees that pneumoconiosis should be ruled out as a cause of the Miner's disability simply because the Miner never received medications for his lung condition. Thus, Dr. Fino's opinion will be given less weight.[10]

Dr. Tuteur prepared a medical report on May 5, 2003. (DX 113) The undersigned finds Dr. Tuteur's report to be well-documented, since it was based on the Miner's autopsy report, numerous test results, and medical reports from other physicians.

Dr. Tuteur determined that, although the Miner was disabled, his disability was not due to his coal mine employment, but rather due to cardiomegaly in association with the advanced coronary artery occlusion. (DX 113)

His findings were clearly outlined and clearly explained. Therefore, the undersigned finds Dr. Tuteur's report to be well-reasoned.

Dr. Farney examined the miner in 1992 and prepared another report on May 14, 2003. (DX 119) While the undersigned finds Dr. Farney's report to be well-documented, it is not found to be well-reasoned. Dr. Farney found the results for the post-exercise 1999 arterial blood gas study to be invalid because the blood sample was drawn from a vein, rather than an artery. (DX 119) This statement is not supported by any evidence in record. Moreover, no other

[10] However, even if the undersigned did not give Dr. Fino's opinion diminished weight, the undersigned's decision would remain unchanged because, even if Dr. Fino's opinion was not given diminished weight, the Employer would not have met its burden. Even with Dr. Fino's opinion receiving full weight, the evidence supports the finding that the Miner was totally disabled due to pneumoconiosis.

physician found this study to be invalid. Since he discredited this 1999 blood gas study, Dr. Farney did not acknowledge that the Miner suffered from hypoxemia, which is a condition that could be related to coal dust exposure. It is also a condition that all other physicians admitted that the Miner had. Thus, Dr. Farney's report will be given less weight.

Dr. Tomashefski prepared a medical report on November 5, 2004. (EX 1) The undersigned finds this report, which was based upon the Miner's autopsy report, autopsy slides, numerous test results and other physicians' reports, to be well-documented.

Dr. Tomashefski concluded that the Miner's oxygen desaturation on exercise was not due to intrinsic lung disease, but rather the result of cardiac dysfunction. (EX 1) He stated that the Miner's pneumoconiosis was too mild to have caused any "respiratory symptoms or respiratory impairment." (EX 1)

His findings were clearly outlined and clearly explained. Thus, the undersigned also finds Dr. Tomashefski's report to be well-reasoned.

In sum, medical reports by Drs. Lincoln, Farney, Poitras, Perper, Caffrey, Naeye, Cander, Fino, Tuteur and Tomashefski were submitted. The reports of Drs. Lincoln, Farney, Caffrey, Naeye, and Fino were given diminished weight, for the reasons enumerated above. The opinions of Drs. Poitras, Perper, Cander, Tuteur and Tomashefski were not given diminished weight.

When examining whether the Miner's total disability was caused by pneumoconiosis, the undersigned finds that Dr. Cander had superior qualifications. Dr. Cander is board-certified in internal medicine. (DX 59) He is the Chairman of the Department of Medicine at the Albert Einstein Medical Center. (DX 59) He is also a professor at Jefferson Medical College. (DX 59) He has received several public appointments to committees that have examined coal workers' pneumoconiosis, including an appointment to Governor William Scranton's Task Force on Coal Workers' Pneumoconiosis. (DX 59) He was also a consultant to the Department of Labor on Coal Workers' Pneumoconiosis. (DX 59) He has also given testimony before the West Virginia Legislature and the Senate Committee on Labor and Welfare on coal workers' pneumoconiosis. (DX 59) He has also published a substantial number of papers. (DX 59)

The undersigned further finds that, while Drs. Perper, Tuteur and Tomashefski are not as qualified as Dr. Cander, they are all highly qualified. However, when examining whether the Miner's total disability was caused by pneumoconiosis, the undersigned finds that Dr. Tuteur, who is board-certified in internal medicine and has had significant experience with pulmonary diseases, is more highly qualified than Dr. Perper or Dr. Tomashefski. Dr. Tuteur is currently an associate physician at Barnes-Jewish Hospital in St. Louis, Missouri. He is also a Director of the Pulmonary Function Laboratory at Washington University School of Medicine. (DX 113) Dr. Tuteur is certified by the American Board of Internal Medicine in internal medicine and pulmonary disease. (DX 113) He has published an extensive number of articles.

The undersigned finds that Drs. Perper and Tomashefski are equally qualified. Dr. Perper has had significant experience in the fields of anatomical and surgical pathology and forensic pathology. (DX34) He is currently the Chief Medical Examiner in Broward County, Florida.

(DX 34) He has also had numerous academic appointments, including the Clinical Professor of Pathology at the University of Pittsburgh and the University of Miami. (DX 34) He has also been a member of numerous medical committees and has published extensively. (DX 34) Dr. Tomashefski is certified by the American Board of Pathology in anatomic and clinical pathology. (EX 1) He teaches Pathology, is a lecturer in Pulmonary Pathology and is a Coordinator of the Pulmonary Pathology Laboratories at Case Western Reserve University. (EX 1) He has also published extensively. (EX 1)

The qualifications of Dr. Poitras are unknown, as his curriculum vitae is not a part of the record.

Drs. Poitras, Perper and Cander concluded that the Miner was totally disabled and that his total disability was due to pneumoconiosis. Drs. Tuteur and Tomashefski concluded that the Miner was not totally disabled due to pneumoconiosis. Dr. Cander's qualifications were deemed to be superior to the qualifications of the other physicians. It is noted that, since this is a request for modification, the burden is placed on the Employer to prove that there was a mistake of fact. Even though the opinions of Dr. Tuteur and Dr. Tomashefski were given full weight, the weight of the new evidence, considered in conjunction with the old evidence, is insufficient to establish that an error was made in the determination that the Miner was totally disabled due to pneumoconiosis.

Was There a Mistake of Fact in the Survivor's Claim?

Since the survivor's claim was filed on August 31, 1999, it is governed by the amended Regulations at 20 C.F.R. Part 718. The Regulations state that "benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. In order to receive benefits, the claimant must prove that (1) The miner had pneumoconiosis; (2) The miner's pneumoconiosis arose out of coal mine employment; and (3) The miner's death was due to pneumoconiosis. . . ." 20 C.F.R. § 718.205(a) (2003).

A person filing a claim after January 1, 1982 is not entitled to the presumptions set forth in §§718.303 or 718.305. Furthermore, a person filing a claim after June 30, 1982 is not entitled to the presumption set forth at § 718.306. However, a Claimant who is not entitled to the presumptions found at §§718.303, 718.305, or 718.306 can still establish entitlement to benefits if she successfully satisfies any of the criteria for proving death due to pneumoconiosis found in § 718.205(c). Subsection 718.205(c)(1) provides that death will be considered to be due to pneumoconiosis where competent medical evidence establishes that the miner's death was due to pneumoconiosis. Subsection 718.205(c)(2) states that death due to pneumoconiosis may be established if pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or if the death was caused by complications of pneumoconiosis. The Tenth Circuit has held that pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death to any degree. See Northern Coal Co. v. Director, OWCP, 100 F.3d 871, 874 (10th Cir. 1996).

The 1992 report by Dr. Farney and the reports by Dr. Lincoln and Dr. Poitras were made prior to the Miner's death. Obviously, none of these reports discuss the cause of the Miner's

death. Therefore, these reports cannot be used to either establish or contradict the idea that the Miner's death was due to pneumoconiosis.

Dr. Leis performed the Miner's autopsy on June 5, 1999. (DX 5) He found that the cause of death was acute myocardial infarction due to occlusive coronary artery disease. (DX 5) He also noted that coal workers' pneumoconiosis was a significant condition that contributed to the Miner's death. (DX 5) During his deposition, Dr. Leis stated that he did not believe that the pneumoconiosis sped up the Miner's development of cardiovascular disease. (DX 49) He determined that the Miner would have died at the same time in the absence of coal workers' pneumoconiosis. However, he also determined that the Miner's coal workers' pneumoconiosis contributed to his death because the pneumoconiosis caused impairment in the Miner's ability to oxygenate his blood. This increased his susceptibility to heart damage. (DX 49)

The undersigned finds Dr. Leis to be a competent and skilled prosector. Neither party has argued that the clinical autopsy findings are flawed. The undersigned finds that there is no good cause to believe that the prosector's clinical findings are inaccurate or that the condition of the miner was fraudulently represented. For example, the undersigned finds that there is no reason to disagree that the Miner's lungs had black anthracotic pigment trapped in the tissue or that the Miner had occlusive coronary artery disease. However, the undersigned cannot give substantial weight to Dr. Leis's opinion, which was expressed during his deposition, concerning whether the Miner's death was due to pneumoconiosis. During that deposition, Dr. Leis stated that the Miner's coal workers' pneumoconiosis contributed to his death. (DX 49) However, he also stated that the Miner would have died at the same time in absence of his coal workers' pneumoconiosis. (DX 49) Dr. Leis failed to articulate how these two statements could both be true. The undersigned finds these two statements to be contradictory.[11]

Dr. Perper found that pneumoconiosis was a substantial contributory cause of the Miner's death. He determined that "severe pulmonary dysfunction and hypoxemia … caused, precipitated or aggravated a final incident of myocardial infarction that occurred on the background of severe arterioscelerotic heart disease…." (DX 33) He based this determination on the Miner's severe obstructive and restrictive pulmonary disease, which was evidenced in clinical testing and the autopsy findings. (DX 33) Dr. Perper noted that pneumoconiosis does not speed the development of cardiovascular disease. (CX 1) However, he also noted that speeding the development of cardiovascular disease was not the only way to hasten a patient's death. (CX 1) He stated that "the increased susceptibility to heart damage by the coal workers' pneumoconiosis related respiratory impairment is a reasonable and effective hastening mechanism of death." (CX 1) He disagreed with Dr. Leis's finding that the Miner would have died at the same time in the absence of coal workers' pneumoconiosis. (CX 1) The undersigned finds Dr. Perper's opinion to be well-documented and well-reasoned.

[11] It is noted that many of the physicians who provided medical opinions concerning whether the Miner's death was due to pneumoconiosis reviewed Dr. Leis's autopsy report and deposition. Even though the undersigned finds that Dr. Leis's opinion regarding the role that pneumoconiosis played in the Miner's death deserves diminished weight, none of opinions of the other the physicians who may have relied, in part, on Dr. Leis's opinion are given less weight for relying on Dr. Leis's opinion because all of these physicians also relied on other evidence that was sufficient to reach a well-documented and well-reasoned conclusion.

Dr. Cander stated that the Miner had pulmonary problems that represented serious and independent risk factors for the sudden spontaneous development of life-threatening cardiac rhythm disturbances. (DX 59) He found that the Miner's death was due to cardiac arrhythmia, which was at least partly related to the Miner's hypoxemia and pulmonary hypertension. (DX 59) In sum, Dr. Cander determined that the Miner's "pneumoconiosis contributed significantly as a cause of his death and hastened it." (DX 59) The undersigned finds Dr. Cander's opinion to be well-documented and well-reasoned.

Dr. Fino's conclusion that the Miner's total disability was not due to pneumoconiosis was given diminished weight because Dr. Fino based this finding on the idea that a person who truly had severe lung disease due to smoke or coal mine dust would have attempted to treat his condition with medications. (EX 3) The undersigned found this logic to be faulty. However, Dr. Fino based his determination that the Miner's death was not due to pneumoconiosis on entirely different evidence and never used the faulty logic described above when concluding that the Miner's death was caused by conditions that were unrelated to pneumoconiosis. (EX 3) Dr. Fino concluded, based on the autopsy, that the Miner died of an acute myocardial infarction. (EX 3) He found that there was no causal relationship between coal dust inhalation or chronic lung disease and death from coronary artery disease. (EX 3) Thus, Dr. Fino determined that there was no evidence that the Miner's lung disease contributed to his death. (EX 3) The undersigned finds Dr. Fino's opinion that the Miner's death was not hastened by pneumoconiosis to be well-documented and well-reasoned.

Dr. Tuteur concluded that the Miner's pneumoconiosis "did not produce symptoms of physiologic impairment. Therefore, coal workers' pneumoconiosis was not a contributing cause to death by myocardial infarction. Neither the inhalation of coal mine dust nor the development of pneumoconiosis hastened his death." (DX 113) Dr. Tuteur noted that, even if he had never worked as a coal miner, the Miner's death would have occurred at the same time. (DX 113) The undersigned finds this opinion to be well-documented and well-reasoned.

Dr. Tomashefski determined that the Miner's death was due to "a massive myocardial infarct, secondary to advanced coronary atherosclerosis." (EX 1) He stated that the Miner's pneumoconiosis was too mild to have caused, or contributed to, the Miner's death. (EX 1) The undersigned finds this opinion to be well-documented and well-reasoned.

Dr. Naeye found that the Miner's total disability and death were due to "[m]yocardiac malfunction due to severe coronary arteriosclerosis, damage to the microcirculation of his heart by cigarette smoking and at the end a coronary artery thrombosis," rather than due to coal workers' pneumoconiosis. (DX 49, DX 60, DX 72) The undersigned finds Dr. Naeye's opinion that the Miner's death was not due to pneumoconiosis is entitled to diminished probative weight, for the same reasons that Dr. Naeye's opinion that the Miner's total disability was not due to pneumoconiosis was given diminished weight. The February 15, 2000 opinion by Dr. Caffrey that the Miner's death was not due to pneumoconiosis is also entitled to diminished probative weight, for the same reasons that Dr. Caffrey's opinion that the Miner's total disability was not due to pneumoconiosis was given diminished weight. Dr. Farney's report is also given diminished weight. Dr. Farney failed to determine that the Miner had hypoxemia, which other physicians' have considered to be a disease that hastened the Miner's death. Since Dr. Farney

did not have a complete picture of the Miner's pulmonary health at the time of his death, Dr. Farney's opinion will be given diminished weight.

In sum, medical reports by Drs. Lincoln, Farney, Poitras, Leis, Perper, Caffrey, Naeye, Cander, Fino, Tuteur and Tomashefski were submitted. Dr. Farney, in his 1992 report, and Drs. Lincoln and Poitras, provide no opinions regarding whether the Miner's death was due to pneumoconiosis. The opinions of Drs. Naeye, Caffrey and Farney are given diminished probative weight. The contradictory conclusions reached by Dr. Leis in his deposition are also found to deserve diminished probative weight. The opinions of Drs. Perper, Cander, Fino, Tuteur and Tomashefski were not given diminished weight.

The opinions of Drs. Perper, Cander, Fino, Tuteur and Tomashefski are conflicting. Drs. Perper and Cander believed that the Miner's death was due to pneumoconiosis. The other doctors disagreed. It is within the province of an administrative law judge to weigh conflicting medical evidence. Hansen v. Director, OWCP, 984 F.2d 364, 368 (10th Cir. 1993). The Tenth Circuit has further stated that, "where medical professionals are in disagreement, the trier of fact is in a unique position to determine credibility and weigh the evidence." Id.at 370.

The undersigned finds that the opinions of Drs. Perper, Cander, Fino, Tuteur and Tomashefski are well-documented and well-reasoned. However, the undersigned further finds some of the physicians to be better-qualified than others. When examining whether the Miner's death was due to pneumoconiosis, the undersigned finds Drs. Perper and Tomashefski, who have backgrounds in pathology, to be more qualified that the other physicians. The undersigned further finds that Drs. Perper and Tomashefski both have significant experience in their field of expertise. They are deemed to be equally qualified. Next, the undersigned finds that Dr. Cander is more qualified than Drs. Fino and Tuteur. All three of these physicians have great experience working with internal medicine and pulmonary diseases. However, only Dr. Cander's curriculum vitae noted substantial experience with coal workers' pneumoconiosis, as well as experience with internal medicine. Dr. Fino, who has published several papers, been appointed to the Division of Pulmonary Disease at St. Clair Memorial Hospital, and been appointed Assistant Clinical Professor of Medicine for the Division of Pulmonary Disease at the University of Pittsburgh (DX 71), and Dr. Tuteur are deemed to be equally qualified.

In general, although the undersigned found Dr. Tomashefski to be highly-qualified to conclude whether the Miner's death was due to pneumoconiosis, the undersigned finds that, overall, the Claimant's physicians are better qualified than the Employer's physicians. It is again noted that, since this is a request for modification, the burden is placed on the Employer to prove that there was a mistake of fact. Even though the opinions of Dr. Tuteur and Dr. Tomashefski were given full weight, the weight of the new evidence, considered in conjunction with the old evidence, is insufficient to establish that an error was made in the determination that the Miner's death was due to pneumoconiosis.

Whether Reopening the Claims Would Render Justice under the Act

The rationale for allowing modification under the Act is to render justice. O'Keefe v. Aerojet-General Shipyards, 404 U.S. 254, 255-256 (1971). In other words, a modification

should only be granted where doing so would render justice under the Act. See Banks v. Chi. Grain Trimmers Ass'n., 390 U.S. 459, 464 (1968).

The undersigned concludes that there is nothing in the record requiring a finding of mistake of fact. The new evidence, considered in conjunction with the prior evidence, was insufficient to allow a modification of the decision in this case. Since the evidence does not support a modification of the earlier decision, there is no reason to reopen or alter the Claimant's prior award of benefits. The interests of justice are served by permitting the Claimant to continue receiving benefits. In other words, justice under the Act would not be served by reopening these claims and altering the award of benefits since the whole of the evidence does not prove that there was a mistake of fact in either claim.

Attorney's Fees

No award of attorney's fees for services to the Claimant is made herein since no application has been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application and his attention is directed to §§ 725.365 and 725.366 of the Regulations. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. Parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in absence of an approved application.

ORDER

The Employer's Petition for Modification is **DENIED**.

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RICHARD K. MALAMPHY
Administrative Law Judge

RKM/kbe
Newport News, Virginia

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence

establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).